**SHORT COMMUNICATION**

**Relation between Hepatitis B Carrier Status and Antibody against Synthetic *Plasmodium falciparum* Erythrocyte Surface (pf155 – RESA) Antigen**

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A survey on *Plasmodium* infection was carried out in gold mine camps located in the Brazilian Amazon. Antibody against *P. falciparum* ring-infected erythrocyte surface antigen (RESA) was quantified by an enzyme-immunoassay in order to assess *P. falciparum* exposure. Hepatitis B, a common infection in this area, was also investigated by serologic markers. Among 520 sampled subjects, 517 (99.4%) admitted previous symptomatic malaria, 106 (20.4%) had positive thick smears for malaria, 82.9% had HBV markers, and 7.1% were HBsAg positive. Anti-RESA titers was significantly lower in HBV carriers than in people with resolved HBV infection suggesting that the anti-RESA immune response could be suppressed by HBV carrier status. Moreover, immunodeficient responses to both infections may take place in some subjects causing concomitant lower anti-RESA response and incapacity to clear HBV.

Key words: malaria falciparum - malaria antigen - hepatitis B virus - Brazilian Amazon

Falciparum malaria and hepatitis B virus (HBV) infections are quite common in the Amazon Basin. Recent reports have shown that this situation also prevails in the southernmost part of the Brazilian Amazon, corresponding to the State of Mato Grosso (Andrade et al. 1995, Souto et al. 1998).

A cross-sectional survey of *Plasmodium* infection was carried out in inhabitants of gold mine camps located in the county of Apiacás, north of Mato Grosso, as reported elsewhere (Souto et al. 2001). Reactivity to antibody against *P. falciparum* ring-infected erythrocyte surface antigen (RESA) was quantified by an enzyme-immunoassay (EIA) in order to assess *P. falciparum* exposure. Gold miners generally have a high prevalence of malaria and HBV infection and are considered to facilitate the spread of both diseases throughout the region (Andrade et al. 1995, Souto et al. 1998). To investigate any relation between malaria and HBV infection the participants were tested for HBV markers as well. The ethical and methodological aspects of this survey were approved by the Federal University of Mato Grosso Research Ethical Committee. The objectives of the study were explained to all participants and informed consent was obtained.

This study was performed between March and June 1996. There were 16 gold mine camps in the county of Apiacás. Five hundred and twenty out of 569 inhabitants of the camps were interviewed and bled. Male subjects comprised 442 (85%) of the overall sample and their age ranged from 3 to 66 years (mean = 32). Most of them (79.6%) were aged between 20 and 40 years.

Thick blood smears were positive in 106 (20.4%) out of the 520 subjects (*P. falciparum*, 56; *P. vivax*, 47; and *P. malariae* 3); 517 (99.4%) admitted previous symptomatic malarial episodes; 431 (82.9%) had been exposed to HBV; 82.9% had HBV markers, and 7.1% were HBsAg positive.

Antibodies against *P. falciparum* were measured by an EIA using RESA produced by Bachem (California, USA), H6215, Lot 119285. This test was standardised and performed at the Blood Bank of Universidade Federal de Mato Grosso, Cuiabá, Brazil. A synthetic peptide [(CTG-(EENV)4-OH)]2 reproducing one of the repeats present in the RESA molecule was used in EIA. Micro-EIA plates were coated with 100 µl/well of the synthetic peptide (2.5 mg/ml in phosphate-buffered saline – PBS) and incubated overnight at 4°C. After spray-wash with PBS-Tween 20 (0.05% PBS-T), plates were blocked with 5% non-fat powdered milk in PBS-T and incubated for 2 h at 37°C. After washing plasma samples were added (100 µl/well) at a dilution of 1:50. After 1 h incubation at 37°C plates were washed again and peroxidase-conjugated goat anti-human IgG was used to detect bound antibodies. As the chromogenic substrate we used 2,2’-azino-di-(3-ethylbenzthiazoline sulfonate). Optical densities were read at 420 nm.

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37 (7.1%) were HBsAg carriers and 394 had previous infection markers (anti-HBc plus anti-HBs, 297, or anti-HBc as the sole marker, 97).

The mean anti-RESA titers of the overall sample was 0.806, ranging from 0 to 3.256. The mean anti-RESA titers of the HBsAg carriers (0.451) was significantly lower than the mean of the rest of subjects (0.832) (P < 0.01) and this difference remained statistically significant (P < 0.001) after multiple linear regression analysis. These findings suggest that the anti-RESA immune response could be suppressed by HBV carrier status. However, immunodeficient responses to both infections may take place in some subjects causing concomitant lower anti-RESA concentration and incapacity to clear HBV. Further studies concerning relation between \textit{P. falciparum} and HBV infection are needed to assess its potential influence in chronic hepatic disease in tropical areas.

REFERENCES


